

# Ischemic Heart Disease

- **Pathophysiology:**
  1. **Imbalance between coronary oxygen supply and demand**—i.e., insufficient blood flow
  2. **Most common cause is coronary atherosclerotic disease**
  3. **Rupture of a plaque → thrombus formation → acute occlusion**

# Major Modifiable Risk Factors

- **Elevated blood cholesterol:**

1. LDL cholesterol is the most important
2. Other risk factors: low HDL, high cholesterol, hypertriglyceridemia, increased total-to-HDL cholesterol ratio, and increased Lipoprotein (a)

- **Tobacco:**

1. Smokers have twice the risk of a nonsmoker
2. Risk is decreased to that of a nonsmoker after 2 full years of cessation

# Major Modifiable Risk Factors (*cont'd.*)

- **Hypertension:**

1. Risk of a cardiovascular event increases
2. Systolic and diastolic values are of equal importance

- **Physical Inactivity and Exercise:**

1. Moderate exercise protects against IHD and stroke even in the **absence** of risk factors
2. Can help increase HDL, control diabetes and obesity, decrease BP

# Major Modifiable Risk Factors (cont'd.)

- **Obesity:**

1. Losing as little as 10-20 lbs can significantly reduce the risk of IHD if overweight

- **Diabetes Mellitus:**

1. Diabetes is an "IHD equivalent."
2. Even when blood glucose is controlled, patients are still at a high risk for IHD.
3. About **75% of patients with diabetes die** from some form of **cardiovascular disease.**

# Major Uncontrollable Risk Factors

- **Age:**

1. Four out of five people who die of IHD are >65 years old

- **Sex:**

1. Males are at greater risk and develop IHD earlier

- **Heredity:**

1. Very important if premature disease is present



## Clinical Case

A 62-year-old male presents with substernal chest pain with exertion and is relieved by rest. He has been having this on and off for 8 months, and the last episode occurred 3 days ago while he was running to the bus. He has a history of well-controlled diabetes and dyslipidemia. The vital signs, physical exam, and ECG are all normal. An exercise stress test is done and shows a 2-mm ST-segment depression.

## Stable Angina

- Occurs during periods of **increased oxygen demand**: exercise and decreased supply (hypotension and anemia)
- Substernal pressure lasting 5-15 minutes with radiation to the jaw, neck, shoulders, or arms, **relieved by rest**
- Pain is usually **predictable** after a certain amount of **exertion**
- Atypical symptoms occur in the elderly or diabetics (autonomic neuropathy)

## Stable Angina (cont'd.)

- **Physical examination:**
  1. Typically normal
  2. May have + S<sub>4</sub> → stiff ventricle
- **Laboratory studies and diagnosis:**
  1. ECG changes **during an attack**
    - Usually ST-segment depression



# Exercise Stress Test

- **Criteria for a positive stress test:**
  1. >2-mm ST-segment depression  
OR
  2. Hypotension defined as a decrease of >10 mm Hg in systolic pressure

# Exercise Stress Test

- Contraindications:
  1. Patient has suspected cardiac instability
  2. Patient is unable to walk or exercise  
→ chemical stress test with either dipyridamole or dobutamine
  3. Baseline abnormalities (e.g. LBBB) makes interpretation difficult → nuclear stress imaging

# Exercise Stress Test

- **Validity of the test:**
  1. Affected by certain medications:  $\beta$ -blockers and digoxin
  2. Asymptomatic, young females have high false-positive rate
  3. Patients with known coronary artery disease have high false-negative rate

## Clinical Case (*cont'd.*)

**False positive test**

## Other Stress Tests to Be Considered

- **Nuclear Stress Test:**
  1. Radioactive material is used to visualize cardiac tissue perfusion
  2. Abnormal amounts of thallium are noted in areas with ↓ perfusion
  3. Overall higher sensitivity and specificity than exercise testing
  4. Bonus: not affected by baseline ECG abnormalities



## Other Stress Tests to Be Considered (*cont'd.*)

- **Dobutamine or Adenosine Stress Test:**
  1. Used in those unable to exercise
  2. Drugs are used to induce tachycardia, as if the patient was exercising
- **Stress Echocardiogram:**
  1. Combines treadmill stress test with an echocardiogram

# Invasive Testing and Therapeutics

- **Cardiac Catheterization:**

1. Used in patients with stable angina for diagnosis and prognosis/risk stratification
2. Angiography is appropriate when noninvasive testing is contraindicated or inadequate
3. Used after a positive stress test to identify patients for stent placement or bypass surgery

# Treatment of Stable Angina

- Acute → sublingual nitroglycerin
- Long-term →
  1. Long-acting nitrates or  $\beta$ -blockers alone or in combination
  2. Aspirin (unless contraindicated)
  3. Statins
  4. Modification of risk factors should be a priority

## Treatment of Dyslipidemia

- Target for hyperlipidemic patients with CAD:
  1. LDL  $\rightarrow$   $<100$  mg/dL
  2. HDL  $\rightarrow$   $>40$  mg/dL
  3. Triglycerides  $\rightarrow$   $< 150$  mg/dL
- Target for very high-risk hyperlipidemic patients:

## Treatment of Dyslipidemia (*cont'd.*)

- Criteria for being a “very high-risk” patient:
  1. Those with established coronary artery disease ***plus any one of the following:***
    - Multiple major risk factors
    - Metabolic syndrome-associated risk factors
    - Acute coronary syndrome



## Treatment of Dyslipidemia (*cont'd.*)

- First-line treatment → **statins**
- If the patient is intolerant to a prescribed statin → decrease the dose and change to another statin



# CABG and PCI

- **Coronary Artery Bypass Grafting (CABG)**
  1. Useful in either:
    - Left main coronary disease
    - Three-vessel disease and LV dysfunction
    - Symptoms despite medical therapy or severe side effects from therapy
  2. More efficacious in those with diabetes
  3. More beneficial but risky in those with low ejection fractions
- **Percutaneous Coronary Intervention (PCI)**
  1. Stent placement is now the standard of care.


# Etiology

- Etiology may be benign *or* life-threatening
  1. Evaluation should focus on **excluding the most serious conditions first**

# History Taking

- History of chest pain is more useful than the physical exam
  1. Duration
  2. Quality
  3. Location
  4. Radiation
  5. Frequency
  6. Alleviating factors
  7. Precipitating factors (**especially exercise**)

# Clinical Pearls to Remember

- Stable angina and acute coronary syndromes
  1. Character: "tightness," "heaviness," or "pressure"
- Inferior wall ischemia
  1. May present with vagal reflexes: bradycardia, hypotension, and/or dizziness/fainting
- Less likely to be ischemia/infarction
  1. "Sharp" or "knife-like" pain that is easily pin-pointed
  2. Pain is reproduced on palpation or changes in position 



## Clinical Pearls to Remember (*cont'd.*)

- True infarction
  1. Pain lasts >20-30 minutes in duration
- Transient ischemia (angina) or esophageal spasm
  1. **Relieved** by nitroglycerin
- Gastroesophageal reflux disease (GERD)
  1. Can **worsen** with nitroglycerin

## Physical Exam

- “Initial impression” is extremely important
  1. Diaphoresis, tachypnea, and/or anxiousness should alert the physician to a potentially **life-threatening process**
  2. Tachycardia and tachypnea (usually nonspecific) are **always present with pulmonary embolism.**

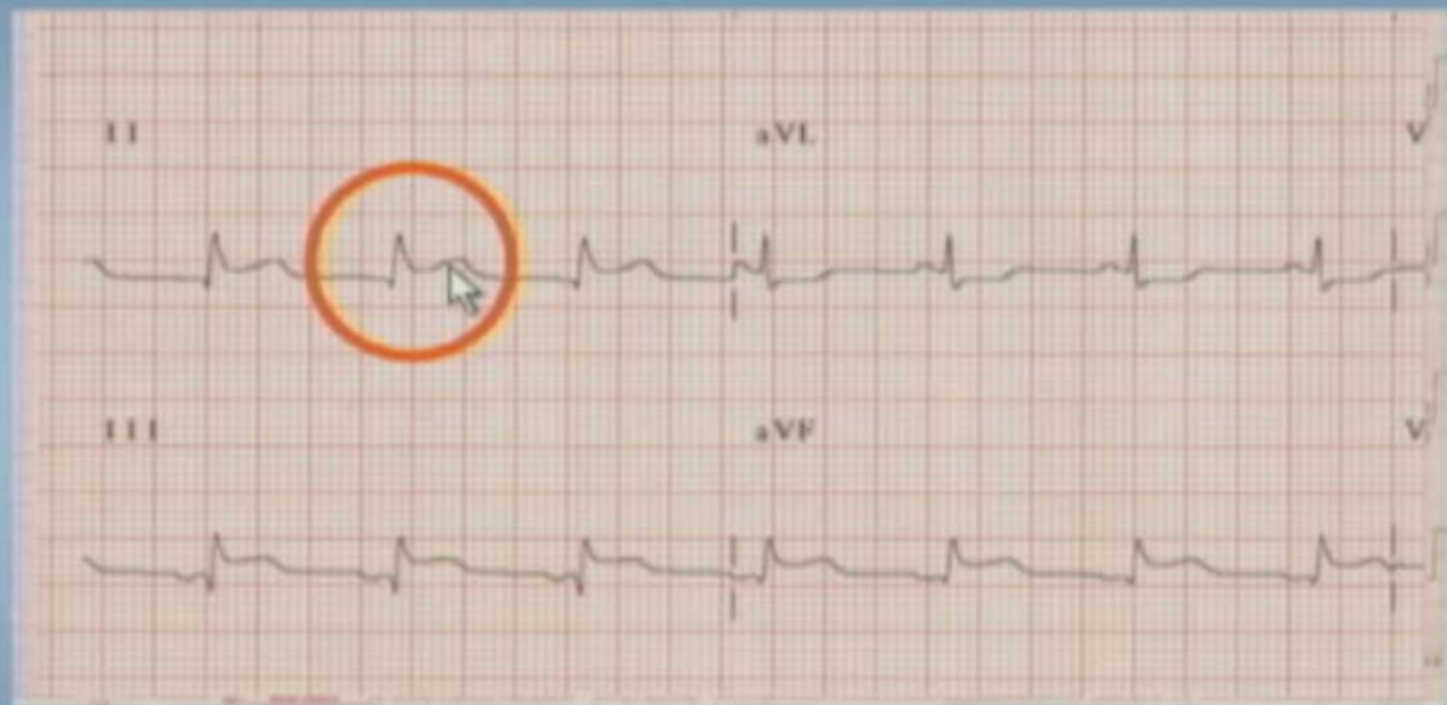
## Physical Exam (*cont'd.*)

- Check for abnormal heart sounds or new onset murmurs. Associations to bear in mind:
  1. Wide split of  $S_2 \rightarrow$  RBBB (right bundle branch block) or right ventricular infarction
  2. New paradoxical split of  $S_2 \rightarrow$  LBBB (left bundle branch block), anterior, or lateral infarction
  3. New  $S_4 \rightarrow$  angina or infarction
  4.  $S_3 \rightarrow$  underlying CHF
  5. New onset aortic regurgitation  $\rightarrow$  50% of patients with aortic dissection
  6. New onset mitral regurgitation  $\rightarrow$  papillary muscle dysfunction

# Testing

- All patients with chest pain should have a 12-lead ECG!!! This is the single most important test for evaluation of cause!!!
- An ECG consistent with myocardial infarction (MI) is the only test required to select patients for emergency reperfusion!!!

# Acute Myocardial Infarction





## Testing (cont'd.)

- Cardiac Biomarkers

1. Cardiac troponins and CK-MB are the two most important markers

- **CK-MB Isozyme** → present **4-6 hours after the onset of ischemia**, peaks in 12-24 hours, and normalizes in 3-4 days. Predicts **early re-infarction**. Often runs in serials every 6-12 hours

## Testing (*cont'd.*)

- Cardiac Biomarkers (*cont'd.*)

1. Cardiac troponins and CK-MB are the two most important markers (*cont'd.*)

- CK-MB Subforms → CK-MB1 found in plasma, CK-MB2 found in myocardial tissue.
  - CK-MB2 >1 U/L and a subform ratio of 1.5 is 96% sensitive and 94% specific

## Testing (cont'd.)

- Cardiac Biomarkers (cont'd.)

1. Cardiac troponins and CK-MB (cont'd.)

- Cardiac Troponins → **preferred markers for diagnosis of myocardial injury**. Troponin is also elevated in renal disease, polymyositis/dermatomyositis
  - Normal CK-MB + ↑ troponin = **minor myocardial damage**
  - ↑ CK-MB + ↑ troponin = **acute myocardial infarction**
  - Remain elevated for weeks → good **late markers of myocardial infarction**

Noncardiovascular Disorders	Differentiating Features
Costochondritis	Pain exacerbated with inspiration; <b><i>reproduced with chest wall palpation</i></b>
GERD	<b><i>Acid reflux</i></b> ; relief with antacids
Peptic ulcer	Epigastric pain
Gallbladder disease	<b><i>RUQ</i></b> abdominal pain and tenderness

Cardiovascular Disorders	Differentiating Features
Myocardial Infarction	Pain more <b>severe</b> , usually <b>&gt;20 minutes in duration</b>
Aortic Stenosis	Typical <b>systolic ejection murmur</b>
Myocarditis	Pain is <b>vague and mild</b>
Pericarditis	Pain is <b>sharp, worse with lying down, relieved by sitting up</b>
Dissecting aortic aneurysm	Pain is <b>sharp, tearing</b> , usually in the <b>back</b>
Mitral valve prolapse	<b>Transient</b> pain, <b>midsystolic click</b> , <b>young female</b> with <b>no risks</b>



<b>Pulmonary Disorders</b>	<b>Differentiating Features</b>
Pulmonary embolus/infarction	<b><i>Tachypnea, dyspnea,</i></b> cough, pleuritic pain, hemoptysis
Pneumothorax	<b><i>Sudden</i></b> onset of <b><i>pain + dyspnea</i></b>



# Causes of Chest Pain

- *Aortic Dissection:*
  1. Severe, sharp, tearing chest pain radiating to the back
  2. Loss of pulses and/or aortic insufficiency
  3. Widened mediastinum on chest x-ray



# Causes of Chest Pain

- **Aortic Dissection:**

1. Severe, sharp, tearing chest pain radiating to the back
2. Loss of pulses and/or aortic insufficiency
3. Widened mediastinum on chest x-ray
4. MI may occur if dissection involves the coronary artery
5. Diagnosis is confirmed with MRI, CT scan, TEE (transesophageal echocardiogram), or aortography

## Causes of Chest Pain (cont'd.)

- **Pulmonary Embolism:**

1. Sudden onset dyspnea, tachypnea, and hypoxemia is prominent
2. Pleuritic pain, especially with pulmonary infarction
3. ECG is usually nonspecific
4. Diagnosis is confirmed with spiral chest CT scan, lung scan, or pulmonary angiogram.

## Causes of Chest Pain (cont'd.)

- **Pericarditis:**

1. May be preceded by a viral illness
2. Pain is sharp, positional, pleuritic, and relieved by leaning forward
3. Pericardial friction rub
4. Diffuse ST elevation without Q-waves
5. Creatinine kinase usually normal
6. Responds to NSAIDs

## Causes of Chest Pain (cont'd.)

- **Myocarditis:**

1. May be preceded by a viral illness (most common etiology in the U.S.). Highly associated with **Coxsackie B virus**. Most common worldwide cause is diphtheria. Most common cause in South Americans is **Chagas disease**
2. Pain is usually vague and mild.
3. Total CK and CK-MB are often elevated
4. +/- conduction abnormalities, and sometimes Q-waves



## Causes of Chest Pain (*cont'd.*)

- **Pneumothorax:**

1. Abrupt onset with sharp pleuritic chest pain and dyspnea; breath sounds absent
2. Confirm with chest x-ray

- **Pleuritis:**

1. Sharp pain increases with inspiration
2. Friction rub or dullness



## Acute Coronary Syndrome

Acute Coronary Syndrome is used to describe a range of thrombotic syndromes. What are they?

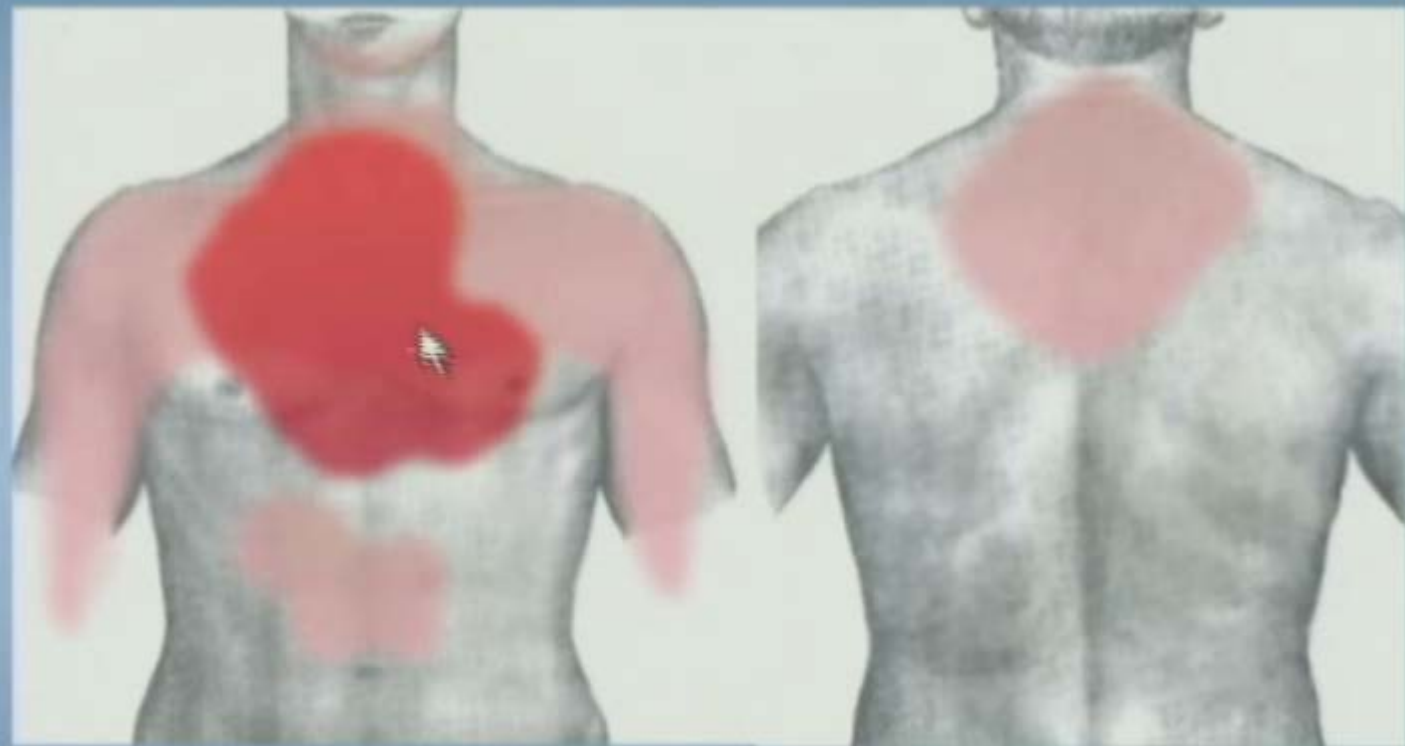
**1. Unstable angina**

**2. Non-ST elevation myocardial infarction (NSTEMI)**

## Acute Coronary Syndrome: Overview

- The initial presentation and management of unstable angina, NSTEMI, and STEMI are very much the same
- Important to distinguish ACS from stable angina

## Location of Pain Associated With ACS



These images were reproduced from Wikipedia,  
<http://www.wikipedia.com>

## Unstable Angina and NSTEMI

Symptoms likely during ACS



Cardiac markers



Normal markers 6-24  
hours after onset

Positive elevation in  
troponin I, troponin T,  
and CK-MB



*Unstable angina*

# Unstable Angina and NSTEMI (cont'd.)

- High-risk features:
  1. Repetitive or prolonged chest pain (>10 minutes)
  2. Elevated cardiac markers
  3. Persistent ECG changes
  4. Hemodynamic instability
  5. Sustained ventricular tachycardia
  6. Syncope
  7. Left ventricular ejection fraction less than 40%
  8. Prior PTCA or CABG
  9. Diabetes
  10. Chronic renal disease



# Unstable Angina and NSTEMI (*cont'd.*)

- **Management:**

1. Thrombolytic therapy is NOT effective!! Infarcted artery is NOT occluded in 60-85% of cases

# Unstable Angina and NSTEMI

## (*cont'd.*)

- Management (cont'd.):
  3. Antiplatelet therapy: aspirin + clopidogrel ***unless contraindications exist***
  4. Antithrombin therapy: unfractionated heparin or subcutaneous enoxaparin for 48-72 hours or until angiography is performed
  5. Glycoprotein IIb/IIIa inhibitors (tirofiban or eptifibatide): especially in high-risk patients
  6.  $\beta$ -blockers unless contraindicated
  7. Intravenous nitroglycerin for refractory pain

## Unstable Angina and NSTEMI (*cont'd.*)

- **Invasive Management:**
  1. Early coronary angiography (within 48 hours) and revascularization is recommended in patients with severe comorbidity

**STEMI**  
**(Formerly Known as Q-wave MI)**

# STEMI

- Symptoms:

1. Substernal pain/pressure with radiation to the jaw, neck, shoulders, or arms, lasting >20 minutes and not relieved completely with nitroglycerin
2. Dizziness, nausea/vomiting, diaphoresis, or shortness of breath
3. Elderly or diabetics often atypical
4. Physical findings are variable depending on infarction area and size

## STEMI (*cont'd.*)

- **Diagnosis:**

1. Clinical symptoms

*Plus the following ECG Changes:*

- Persistent ST-segment elevation of  $>1$  mm in two contiguous leads





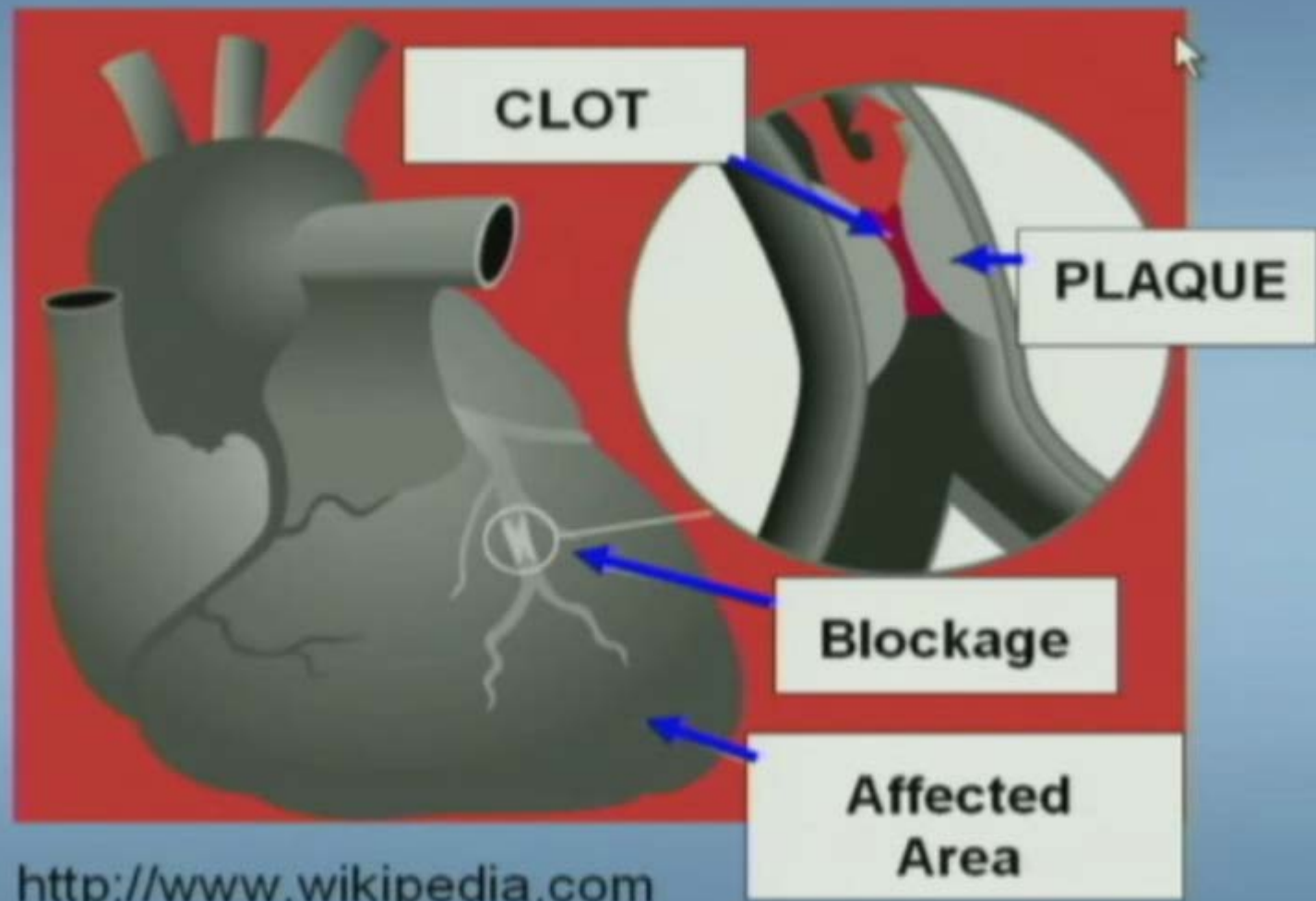
## STEMI (*cont'd.*)

**This means that you do not initially need elevated biochemical markers for the diagnosis of STEMI!**

Area Infarcted	ECG Changes	Artery
Inferior	II, III, aVF	RCA
Anteroseptal	V <sub>1</sub> -V <sub>3</sub>	LAD
Anterior	V <sub>2</sub> -V <sub>4</sub>	LAD
Lateral	I, aVL, V <sub>4</sub> , V <sub>5</sub> , and V <sub>6</sub>	LAD or circumflex
Posterior	V <sub>1</sub> -V <sub>2</sub> : Tall, broad initial R-wave; ST depression; tall, upright T-wave; usually occurs in association with inferior or lateral MI	

**Table I-6-6: ECG Evolution of MI**

<b>ECG Change</b>	<b>Onset</b>	<b>Disappears</b>
Hyperacute T-waves (tall, peaked T-waves in leads facing the infarct)	Immediate	6-24 hours
ST segment elevation	Immediate	1-6 weeks
Q-waves >0.04 seconds	One-several days	Years to never
T-wave inversion	6-24 hours	Months to years



# Emergent Reperfusion Therapy

## (cont'd.)

- Choice between PCI versus thrombolysis (cont'd.)
  3. Where PCI is delayed or not available, reperfusion with thrombolysis is best
  4. Greatest benefit is to those with **anterior infarction**, ST-elevation, or bundle branch block and symptoms for <12 hours
  5. Streptokinase and alteplase → rapid IV infusion
  6. Reteplase and tenecteplase → rapid bolus injection



## Emergent Reperfusion Therapy (*cont'd.*)

- *The bottom line:*
  1. Consider thrombolytic therapy as an alternative to primary PCI in suitable candidates with:
    - ST-elevation MI ( $>1$  mm ST-elevation in two contiguous leads)
    - New LBBB (left bundle branch block)




## Contraindications to Thrombolytics

- **Absolute Contraindications:**
  1. Active bleeding or bleeding diathesis
  2. Significant closed head or facial trauma within 3 months
  3. Suspected aortic dissection
  4. Prior intracranial hemorrhage
  5. Ischemic stroke within three months

## Contraindications to Thrombolytics (*cont'd.*)

- **Relative Contraindications:**

1. Recent major surgery (<3 weeks)
  2. Traumatic or prolonged cardiopulmonary resuscitation
  3. Recent (within 4 weeks) internal bleeding
  4. Active peptic ulcer
  5. Severe, poorly controlled hypertension
  6. Ischemic stroke <3 months
- 

## Late Presentation: 12 Hours After Onset of Symptoms

- Reperfusion and PCI are not indicated in stable patients
- CABG may be considered in those with good anatomy
- CABG is also considered in those with cardiogenic shock or in association with mechanical repair

## Adjuvant Therapy Used With Reperfusion

- *Antiplatelet therapy:*
  1. Aspirin → all patients unless contraindicated
  2. Clopidogrel → all patients undergoing PCI with stent placement and those undergoing fibrinolytic treatment

## Adjuvant Therapy Used With Reperfusion (cont'd.)

- **Glycoprotein IIb/IIIa Inhibitors:**
  1. Primary PCI → early use has greatest benefit
- **Emergency CABG:**
  1. Failed PCI
  2. Persistent or recurrent ischemia refractory to medical therapy

## Other Testing in ACS

- **Exercise ECG Testing (Stress Test):**
  1. Submaximal testing 4-7 days after infarction
  2. Test 3-6 weeks post-infarction maximal
  3. Assesses prognosis and those with reversible ischemic who may need an angiogram and/or CABG
- **Myocardial Perfusion Imaging:**



# Discharge Medications After ACS

- Aspirin → 75-325 mg/day
- Clopidogrel → in intolerant to aspirin or as an alternative for 9-12 months
- $\beta$ -blocker → indefinitely
- ACE inhibitors → added for CHF or left ventricular dysfunction (EF <40%) is present
- Statins → should be started in the hospital
- Nitrates → Short-acting nitrates as a rule. Long-acting only if chest pain is persistent.

## Discharge Medications After ACS (*cont'd.*)

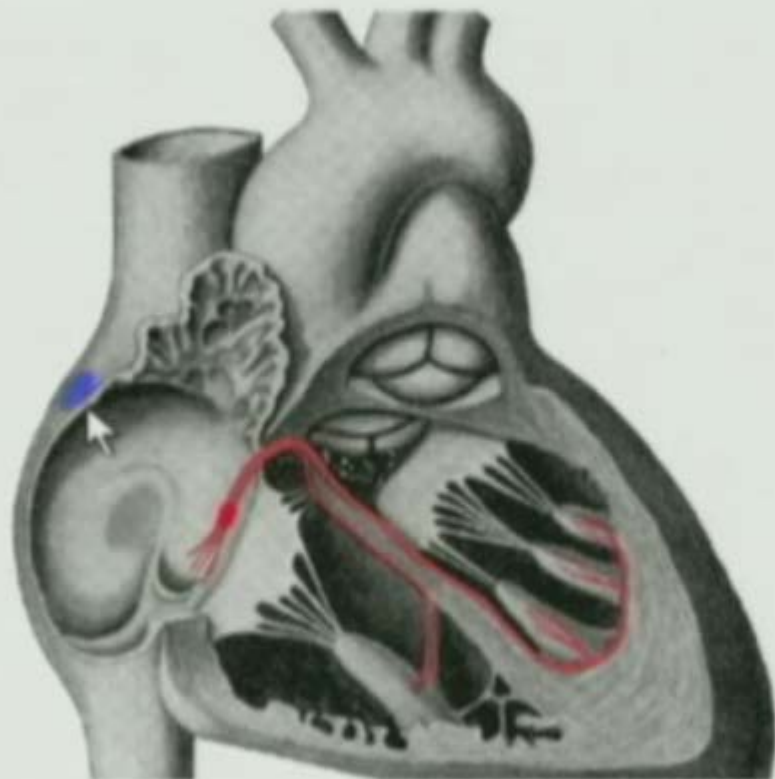
<u>Pneumonic</u>	<u>Significance</u>
<u>A</u>	Aspirin and anti-anginals
<u>B</u>	$\beta$ -blockers and blood pressure
<u>C</u>	Cholesterol and cigarettes
<u>D</u>	Diet and diabetes
<u>E</u>	Education and exercise

# Complications of ACS

# Electric Disturbances

- **Dysrhythmias:**
  1. Bradycardia
  2. Premature beats
  3. Tachyarrhythmias →  
supraventricular or ventricular
- **Conduction Abnormalities:**
  1. Atrioventricular nodal

# Normal Conduction



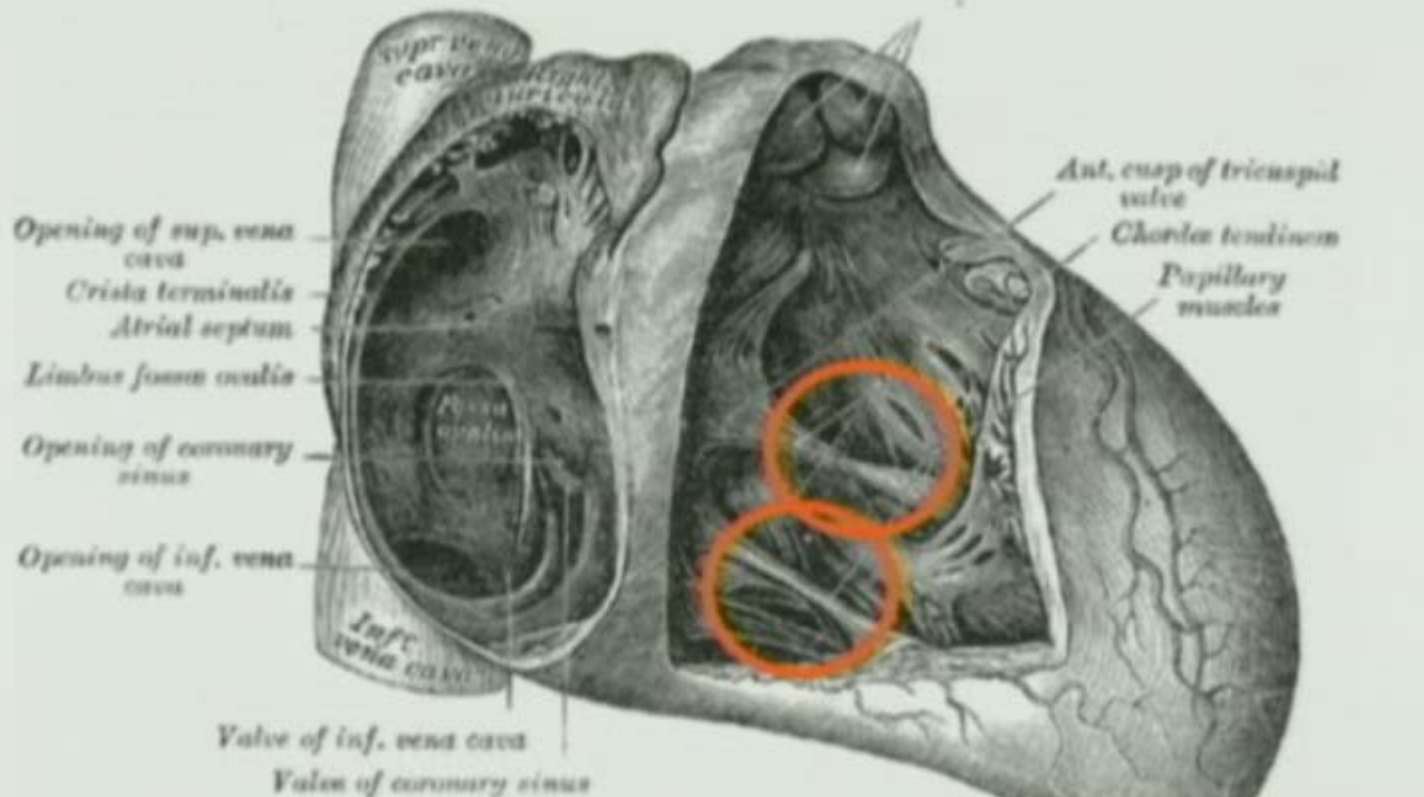
This image was reproduced from Wikipedia,  
<http://www.wikipedia.com>

# Pump Dysfunction

- Contractile dysfunction
- Mechanical disruption
- Electromechanical dissociation



# Papillary Muscle Rupture



**KAPLAN MEDICAL**  
Reproduced from Wikipedia, <http://www.wikipedia.com>

## ACS Complications (*cont'd.*)

- Ischemia
- Pericarditis → Dressler syndrome
  1. Treat with NSAIDs, aspirin, +/- steroids
- Thromboembolic → mural thrombi or deep venous thrombosis

## Prinzmetal Angina

- Secondary to sudden coronary vasospasm
- Associated with ST-segment elevation
- Occurs at rest
- Patients often awaken from sleep with severe chest pain
- Occurs more often in females and those with a history of migraine
- Stress testing and angiography are normal
- Definitive diagnosis → ergonovine during angiography → triggers vasospasm → treatment with calcium-channel blockers or nitrates

## Mitral Stenosis: Etiology

- *Most common lesion due to rheumatic fever*
- Rarely due to a genetic defect
- 2/3 of patients are female

# Mitral Stenosis: Symptoms

- Dyspnea
- Orthopnea/paroxysmal nocturnal dyspnea
- Fatigue
- Wasting
- Hemoptysis (ruptured vessels)
- Systemic embolism (stagnation of left atrial blood)
- Hoarseness (enlarged left atrium on the recurrent laryngeal nerve)
- Right-sided heart failure: hepatomegaly, ascites, peripheral edema

## Mitral Stenosis: Signs

- Atrial fibrillation
- Rales
- Decreased pulse pressure
- Loud  $S_1$
- *Opening snap following  $S_2$*
- *Diastolic rumble (low-pitched, apical murmur)*
- Sternal lift (due to enlarged right ventricle)



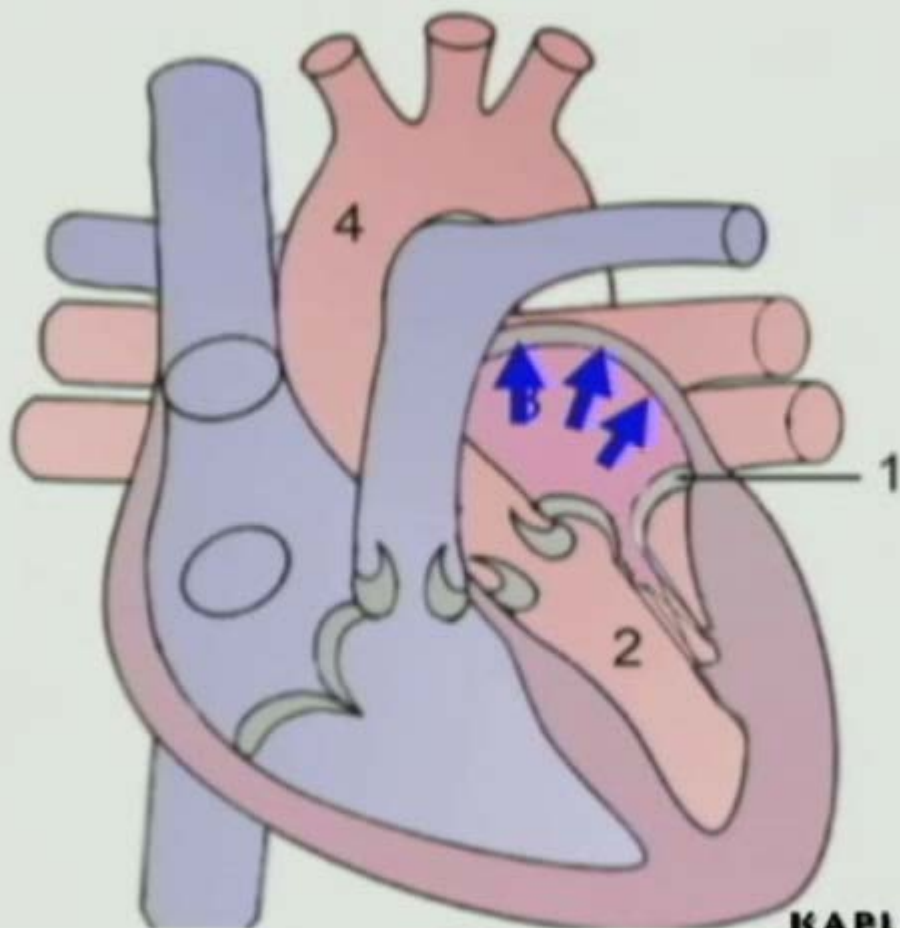
# Mitral Stenosis: Diagnosis

- **EKG:**
  1. May show right ventricular hypertrophy
  2. May show right and left atrial abnormalities
  3. +/- A-fib
- **Chest x-ray:**
  1. Large left atrium, pulmonary hypertension, large pulmonary artery are all possible
- **Echocardiography:**
  1. Thickened mitral valve leaflets and decreased excursion

# Mitral Stenosis: Treatment

- **Medical therapy:**
  1. Diuretics and salt-restricted diet
  2. Digitalis and anticoagulants in A-fib
- **Surgical therapy:**
  1. Balloon valvuloplasty
  2. Mitral commissurotomy or valve replacement
  3. Pulmonary hypertension does not contraindicate surgery

# Mitral Regurgitation



**KAPLAN** MEDICAL

This image was reproduced from Wikipedia, <http://www.wikipedia.com>

# Mitral Regurgitation: Etiology

- Due to abnormalities in the mitral valve leaflets, annulus, and cordae tendinae
  1. Common cause is dilation of the left ventricle
  2. Men > women

# Mitral Regurgitation: Diagnosis

- **Physical Signs:**

1. Hyperdynamic, displaced apical impulse
2. Carotid upstroke decreased but brisk
3. Holosystolic apical murmur radiating to the axilla +/- thrill
4.  $S_3$  usually heard with a soft  $S_1$  and widely split  $S_2$
5. Distended neck veins



## Mitral Regurgitation: Diagnosis (*cont'd.*)

- *EKG* → left-ventricular hypertrophy and left atrial enlargement
- *Chest x-ray* → cardiac enlargement with vascular congestion
- *Echocardiography* → left atrial and/or left ventricular dilation

# Mitral Regurgitation: Treatment

- Medical Therapy:

1. Increase cardiac output and relieve pulmonary congestion
  - Arteriolar vasodilators
  - Digitalis
  - Diuretics
  - Anticoagulants

- Surgery:

1. Guidelines for selecting patients:
  - With significantly limiting symptoms and severe regurgitation
  - Symptoms persist despite optimal medical therapy

# Mitral Valve Prolapse

- **Most common congenital valvular lesion**
- More common in females and those with connective tissue disease
  1. Marfan syndrome
- **Presentation:**
  1. Usually asymptomatic unless arrhythmias occur: chest pain, lightheadedness, palpitations, and syncope



# Mitral Valve Prolapse (cont'd.)

- Auscultation:

1. **Mid-to-late systolic click** and late systolic murmur heard best at the apex
2. Worsens with Valsalva maneuver
3. Improves with squatting

- Complications:

1. Serious arrhythmias and sudden death
2. CHF
3. Bacterial endocarditis
4. Calcifications of the valve
5. TIAs

## Mitral Valve Prolapse (*cont'd.*)

- **Labs:**

1. 2-D Echo → systolic displacement of the mitral leaflets

- **Treatment:**

1. Medical management → endocarditis prophylaxis if a murmur is present,  $\beta$ -blocker for chest pain/arrhythmia



# Aortic Stenosis



# Aortic Stenosis: Pathophysiology

- Calcification and degeneration of a congenitally normal valve, most commonly in the elderly
- Calcification and fibrosis of congenital bicuspid valve
- Rheumatic valvular disease



## Aortic Stenosis: Symptoms

- Angina, syncope, and dyspnea
- Pulsus tardus et parvus
- Carotid thrill
- Systolic ejection murmur heard best at right sternal border, with a thrill, harsh quality, and radiates to the carotids
- S<sub>4</sub> gallop
- A<sub>2</sub> decreased, S<sub>2</sub> single or paradoxical split

## Aortic Stenosis (cont'd.)

- Diagnosis:

1. ECG: LV hypertrophy
2. Chest x-ray: calcifications, cardiomegaly, pulmonary congestion
3. Echocardiography: thick aortic valve leaflets with decreased excursion and LVH

- Treatment:

1. Patients must receive endocarditis prophylaxis
2. Surgery when valve area is  $\downarrow < 0.8 \text{ cm}^2$
3. Generally, if patients have symptoms, they need surgery
4. Balloon valvuloplasty in those who cannot tolerate surgery

# Aortic Regurgitation

# Aortic Regurgitation

- Most common cause is *systemic hypertension*
- Other causes include:
  1. Infectious endocarditis
  2. Syphilis
  3. Ankylosing spondylitis
  4. Marfan syndrome
  5. Aortic dissection

## Aortic Regurgitation (*cont'd.*)

- **Clinical findings:**

1. Dyspnea most common
2. Diastolic-decrescendo murmur most common
3. Systolic flow murmur
4. Duroziez sign: systolic/diastolic thrill or murmur over the femoral arteries
5.  $S_3$  in early left ventricular decompensation



## Aortic Regurgitation (*cont'd.*)

- **Diagnosis:**

1. ECG: LV hypertrophy
2. Chest x-ray: LV or aortic dilation
3. Echocardiography: Dilated LV and aorta; left-ventricular volume overload; fluttering of anterior mitral leaflet

- **Treatment:**

1. Endocarditis prophylaxis
2. Salt restriction, diuretics, afterload reduction
3. Aortic valve replacement when symptoms are severe or ejection fraction plummets



## Myocardial and Pericardial Disease

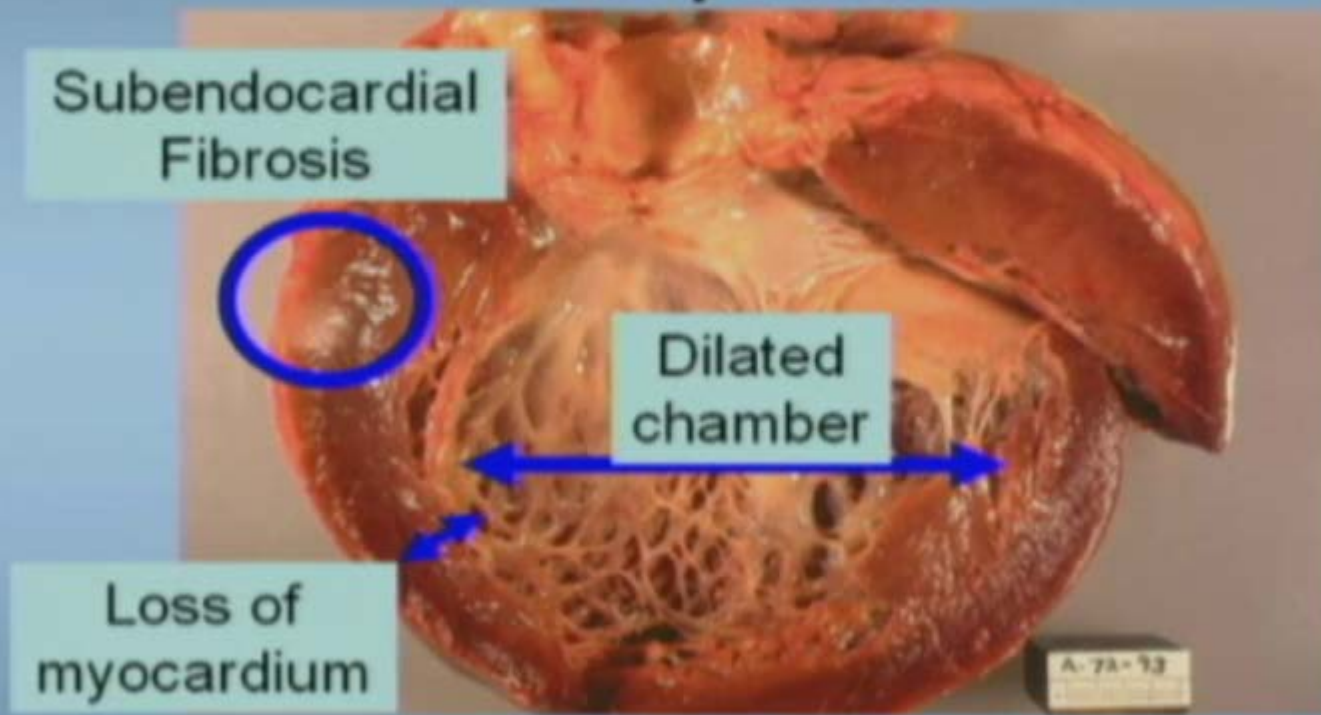
# Cardiomyopathy: Overview

<u>Subtype</u>	<u>Characteristics</u>
Dilated	Biventricular dilation
Hypertrophic	Marked hypertrophy of the left ventricle +/- right ventricle, disproportionate hypertrophy of the septum
Restrictive	Reduced compliance, due to infiltration of the myocardium
Obliterative	Thickened endocardium or mural thrombi, or both, act as space-occupying lesions

# Dilated Cardiomyopathy

- Diminished contractility
- Usually both ventricles
- ***Most common indication for heart transplant***
- **Etiology:**
  1. Ischemic → ***most common cause***
  2. Alcoholism → ***2nd most common cause***
  3. Peripartum
  4. Post-myocarditis
  5. Toxins (lead, cobalt, arsenic)
  6. Doxorubicin hydrochloride, cyclophosphamide, vincristine

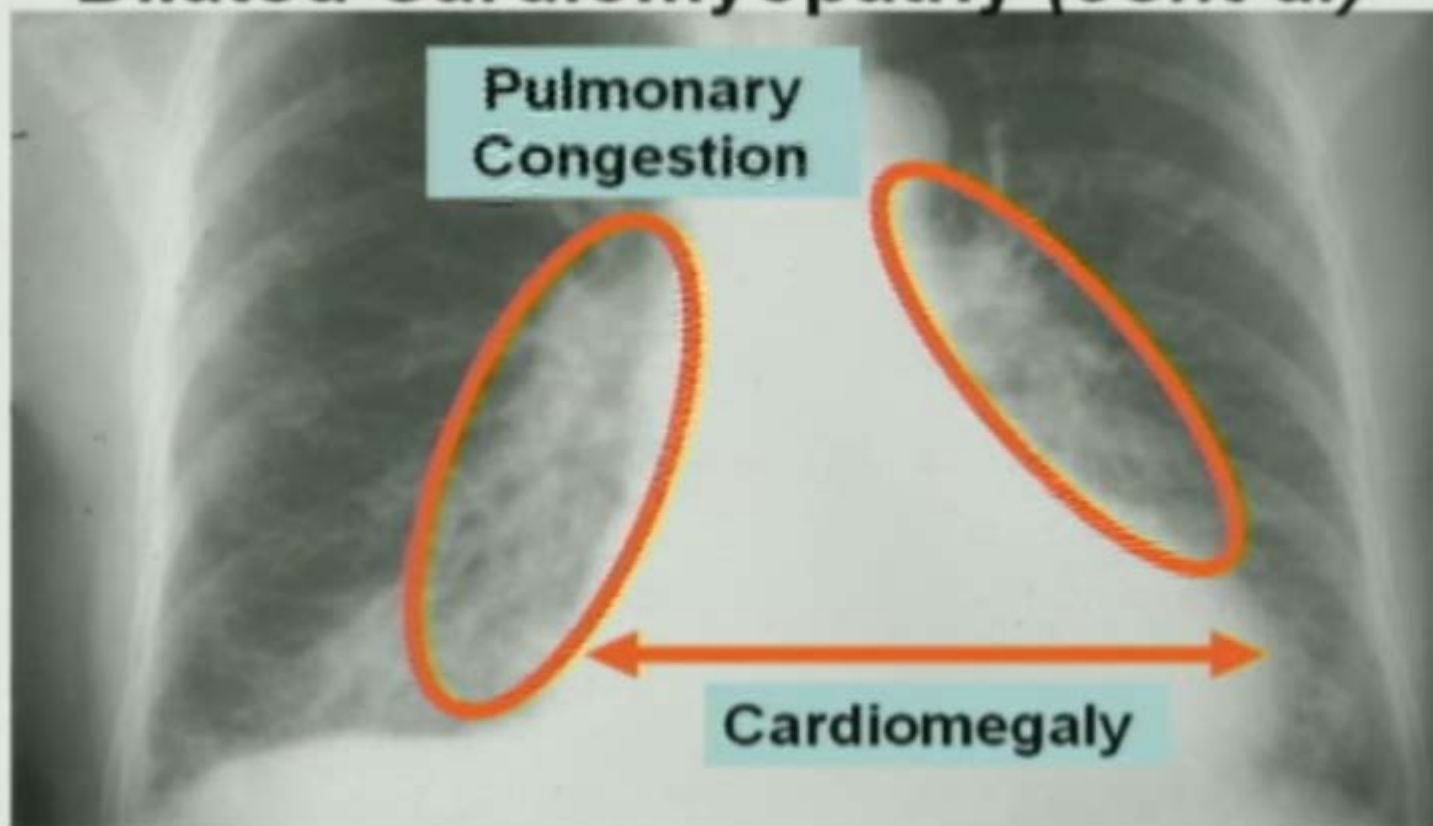
# Dilated Cardiomyopathy: Idiopathic



This image was reproduced from the Public Health Image Library, <http://phil.cdc.gov>.

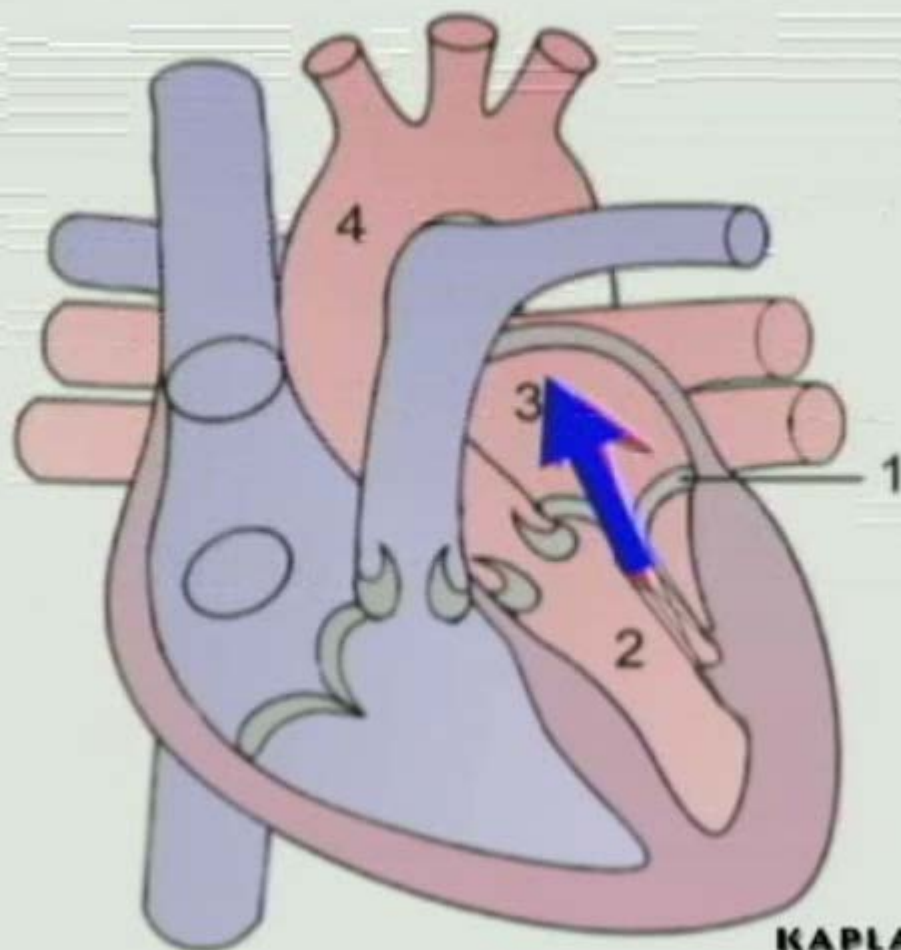


## Dilated Cardiomyopathy (*cont'd.*)



© 2007 Kaplan Inc., Reproduced with permission from DxR Imaging





**KAPLAN** MEDICAL

This image was reproduced from Wikipedia, <http://www.wikipedia.com>

## Factors That Increase Obstruction

<b><i>Mechanism</i></b>	<b><i>Physiologic or Pharmacologic Factors</i></b>
↑ Contractility	<ul style="list-style-type: none"><li>• Cardiac glycosides</li><li>• <math>\beta</math>-agonists</li><li>• Tachycardia</li><li>• Premature beats</li></ul>
↓ Preload	<ul style="list-style-type: none"><li>• Valsalva or standing</li><li>• ↓ Intravascular volume</li><li>• Nitrates and vasodilators</li><li>• Tachycardia</li></ul>
↓ Afterload	<ul style="list-style-type: none"><li>• Hypovolemia</li><li>• Nitrates and vasodilators</li></ul>

## Hypertrophic Obstructive Cardiomyopathy (*cont'd.*)

- **Clinical Manifestations:**

1. Dyspnea
2. Angina/palpitations/presyncope-syncope
3. Palpable S<sub>4</sub> gallop, systolic murmur + thrill, mitral regurgitation murmur
4. Sudden death can be the first manifestation



# Hypertrophic Obstructive Cardiomyopathy (*cont'd.*)

- **Diagnosis:**

1. ECG → Left axis deviation, pseudo Q-waves ( $V_1$ - $V_3$ ), ventricular arrhythmias
2. Echo → **Mainstay of diagnosis**;  
hypertrophy, systolic anterior motion of mitral valve, midsystolic closure of aortic valve

- **Treatment:**

1.  $\beta$ -blockers
2. Calcium channel blockers
3. Disopyramide on occasion
4. Surgery for severe cases

# Restrictive Cardiomyopathy

- Least common cardiomyopathy
- Characterized by *rigid, noncompliant ventricular walls*
- **Etiology:**
  1. Infiltrative:
    - Sarcoidosis, amyloidosis, hemochromatosis, neoplasia

## Restrictive Cardiomyopathy (*cont'd.*)

- **Clinical Manifestations:**
  1. Dyspnea, exercise intolerance, and weakness
  2. Edema, elevated JVP, hepatomegaly, ascites, S<sub>4</sub> and S<sub>3</sub> gallop, Kussmaul sign



# Restrictive Cardiomyopathy (*cont'd.*)

- **Diagnosis:**

1. X-ray → mild cardiomegaly, pulmonary congestion
2. ECG → low voltage, conduction disturbances, Q-waves
3. Echo → rigid myocardium, characteristic seen in amyloidosis with thickening of all cardiac structures

- **Treatment:**

1. No good treatment

# Acute Pericarditis

- **Etiology:**

1. Idiopathic
2. Infections
3. Vasculitis
4. Disorders of metabolism
5. Neoplasms
6. Trauma

## Acute Pericarditis (*cont'd.*)

- **Clinical presentation:**
  1. Substernal-left sternal chest pain worsened with recumbency, coughing, and deep inspiration
  2. Relieved by sitting up or leaning forward
  3. Pericardial friction rub
- **Diagnosis:**
  1. ECG → diffuse ST-segment elevation with upright T-waves

## Acute Pericarditis (*cont'd.*)

- **Differential Diagnosis:**
  1. Acute MI → pericarditis has characteristic ST-segment elevation, **absence of reciprocal leads**, and **no Q-waves**
- **Treatment:**

## Pericardial Effusion

<u>Character</u>	<u>Etiology</u>
Transudate	CHF, fluid overload, hypoproteinemia
Exudate	Pericardial injury
Sero-sanguinous	Tuberculosis, neoplasm
Frank blood	Aortic aneurysm, dissection, blunt/penetrating trauma, bleeding 2° to coagulation defects, myocardial rupture post-MI

**Fluid  
accumulates  
*slowly***



**Pericardial  
expansion and  
accommodation**

**Fluid  
accumulates  
*rapidly***



**Cardiac  
tamponade!!!**



## Pericardial Effusion (cont'd.)

- Diagnosis:

1. Echocardiography →

- Echo-free pericardial space between the posterior pericardium and posterior left ventricular epicardium
- In large effusions, the heart may swing freely, and may be associated with electric alternans

2. Chest x-ray → "water-bottle" configuration of the cardiac silhouette

- Treatment:

# Cardiac Tamponade

- Medical emergency!!!
- Effusion develops rapidly → compression of the heart → rapid heart failure
- **Etiology:**
  1. Neoplasia
  2. Idiopathic (usually viral)
  3. Nonviral infection: tuberculosis and suppurative
  4. Intrapericardial hemorrhage
  5. Wounds: CT surgery
  6. Uremia
  7. Radiation (mediastinal)
  8. Vasculitis

## Cardiac Tamponade (cont'd.)

- Clinical Manifestations:

1. Dyspnea, orthopnea, and fatigue
2. *Pulsus paradoxus*
3. *Neck vein distention with clear lung fields*
4. Shock
5. *Decreased, muffled heart sounds*

- Diagnosis:

1. Echocardiography and cardiac catheterization

- Treatment:

1. Pericardiocentesis
2. Subxiphoid surgical drainage

## Constrictive Pericarditis (*cont'd.*)

- **Etiology:**

1. Idiopathic
2. Secondary to open heart surgery or thoracic radiation
3. Postviral infection

- **Clinical Manifestations:**

1. Dyspnea on exertion (common)
2. Orthopnea
3. S/S of right sided-failure
4. Kussmaul sign
5. Distant heart sounds and a “pericardial knock” (often confused with S<sub>3</sub>)

# Constrictive Pericarditis (*cont'd.*)

- **Diagnosis:**

1. ECG → low voltage and nonspecific T-wave changes
2. Chest x-ray → normal
3. Chest CT or MRI → thickened pericardium, pericardial calcifications if tuberculous

- **Differential Diagnosis:**

1. Restrictive cardiomyopathy

## Constrictive Pericarditis (*cont'd.*)

- **Treatment:**

1. Mild sodium restriction
2. Diuretics
3. Pericardiectomy if required





## Rate and Rhythm Disturbances

# Sinus Bradycardia

- ***Ventricular complexes of normal width, spacing, and rate <60 beats per minute***
- **Etiology:**
  1. Excess vagal tone: acute MI, carotid sinus pressure, vomiting, Valsalva maneuver, phenothiazines, digitalis
  2. Decreased SA node automaticity:  $\beta$ -blockers, CCAs
  3. Marathon running and swimming
  4. Hypothyroidism
  5. Normal variants

## Sinus Bradycardia (*cont'd.*)

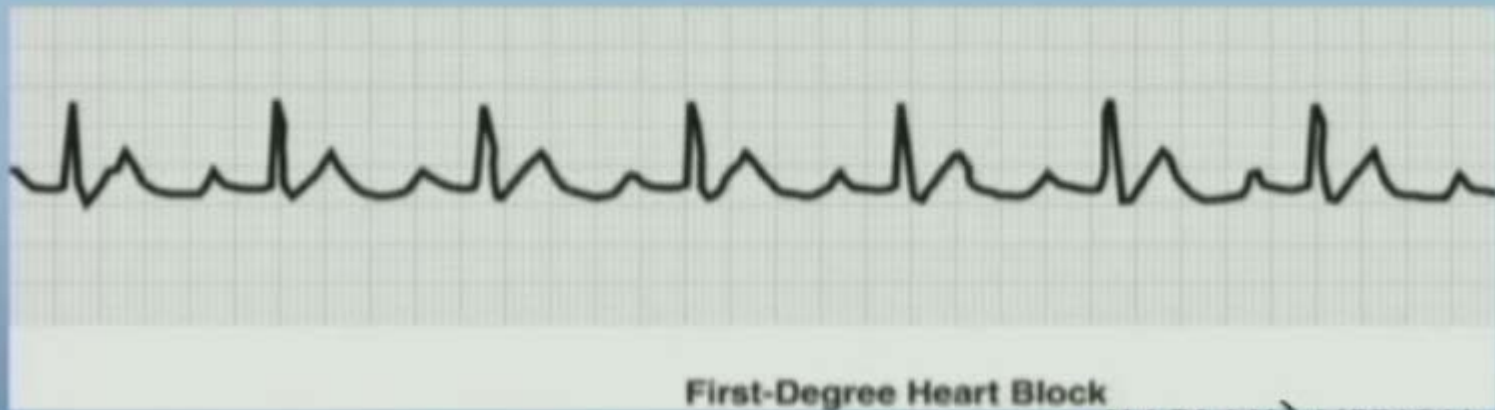
- **Treatment:**
  1. No symptoms = no treatment
  2. Acute symptoms → atropine
  3. Chronic symptoms and bradycardia  
→ pacemaker

# Atrioventricular (AV) Block

- ***Clinical, based on ECG:***
  1. First-degree block
  2. Second-degree block
  3. Third-degree block

## First-Degree Block

- PR interval  $>0.20$  seconds
- Etiology: aging, digitalis, increased vagal tone, ischemia, inflammation, cardiomyopathy



First-Degree Heart Block

## Second-Degree Block

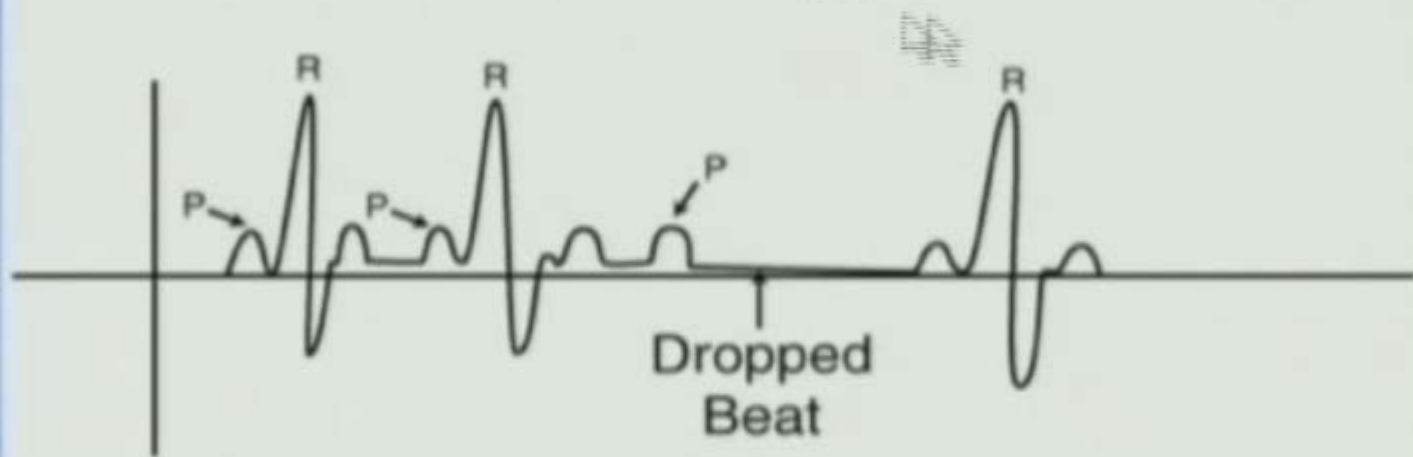
	<u><i>Mobitz Type I (Wenckebach)</i></u>	<u><i>Mobitz Type II</i></u>
<u><i>Site of block</i></u>	AV node	Infranodal
<u><i>ECG</i></u>	PR interval progressively lengthens until beat is "dropped"	PR interval usually of normal duration and consistent



## Mobitz Type I

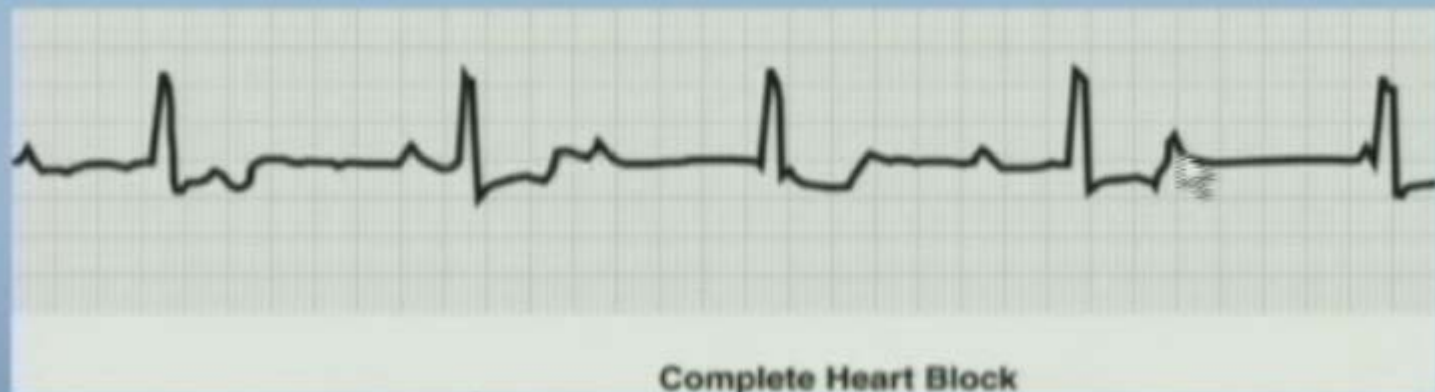


## Mobitz Type II



## Third-Degree Block

- Blockage of all atrial beats; ventricles beat through an escape focus distal to the block



## Third-Degree Block (*cont'd.*)

- **Clinical Manifestations:**

1. Adams-Stoke attacks +/- CHF
2. What is an Adams-Stoke attack???

- **Treatment:**

1. Pacing

# Sinus Tachycardia

- Ventricular complexes are of normal width, evenly spaced, and rate is  $>100$  BPM
- **Etiology:**
  1. Secondary to any condition that increases the heart rate:
    - Fever
    - Hypotension
    - Pain
    - Anxiety
    - Drugs
    - Thyrotoxicosis

## Paroxysmal Supraventricular Tachycardia

- Sudden onset of an ectopic tachyarrhythmia followed by abrupt cessation
- Initiated by a supraventricular premature beat
- Includes paroxysmal atrial tachycardia
- 80% due to re-entry (AV node)
- Rate between 130-200 beats/minute and regular



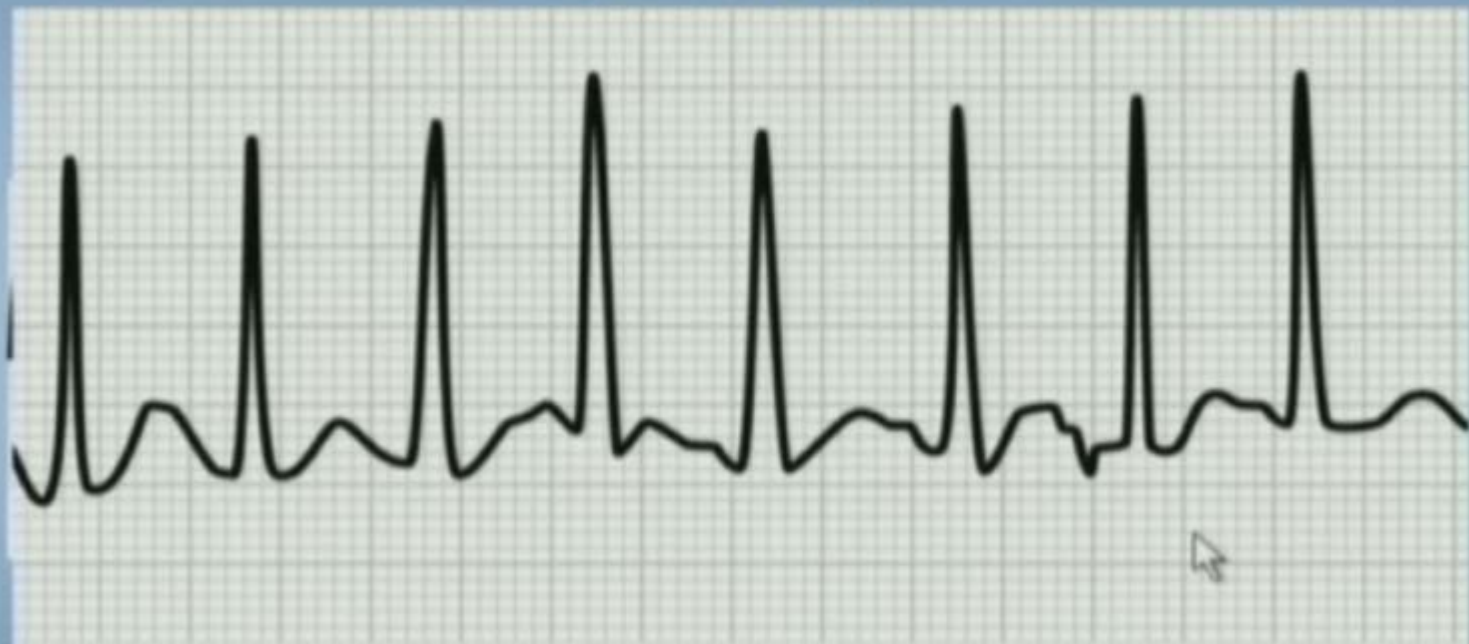
# Paroxysmal Supraventricular Tachycardia (cont'd.)

- Treatment:
  1. Increase vagal tone → carotid sinus massage
  2. Drugs of choice → *IV verapamil or adenosine*
    - Cures 90% of cases
  3. IV propranolol or esmolol
  4. IV digitalis

## Multifocal Atrial Tachycardia

- Irregular, supraventricular rhythm between 100-200 beats/minute
- P-wave morphology and PR-interval ***varies from beat to beat*** (at least 3 to be diagnostic)
- Each ***QRS*** complex is ***preceded*** by a ***P-wave***
- Seen in the elderly and those with respiratory failure

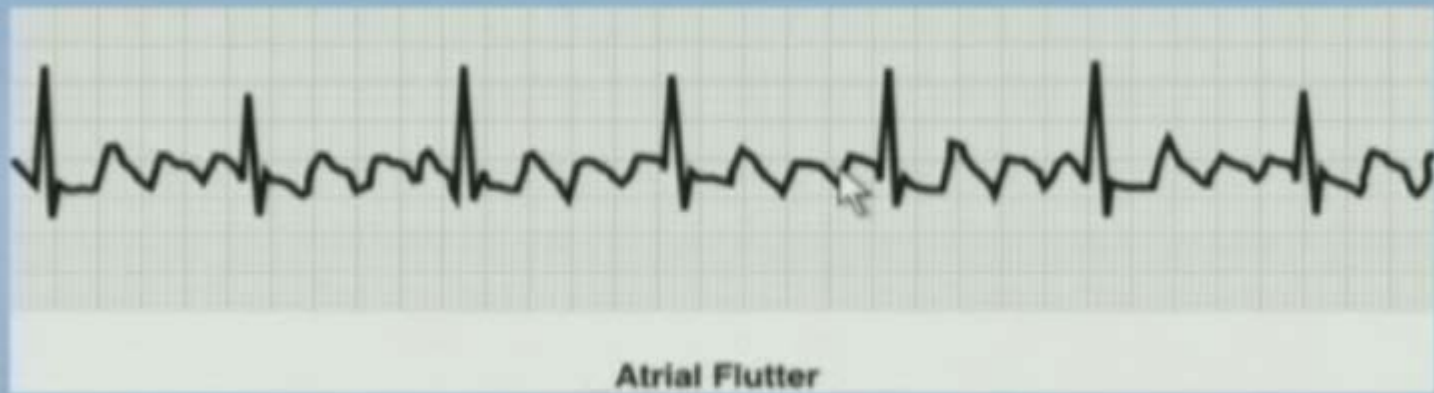
## Multifocal Atrial Tachycardia (*cont'd.*)



**Multifocal Atrial Tachycardia**

© 2001 Kaplan Inc.

## Atrial Flutter (*cont'd.*)



## Atrial Flutter (*cont'd.*)

- **Treatment:**

1. Cardioversion if hemodynamically unstable
2. Digitalis
3. Verapamil, diltiazem
4.  $\beta$ -blockers

# Atrial Fibrillation

- **Etiology/Pathophysiology:**
  1. Supraventricular tachyarrhythmia
  2. Uncoordinated atrial activation
  3. Decline in atrial function
  4. P-waves are replaced by fibrillatory waves
  5. Rapid, irregular ventricular response occurs but is dependant on several factors:
    - Electrophysiology of the AV node
    - Level of sympathetic and parasympathetic tone



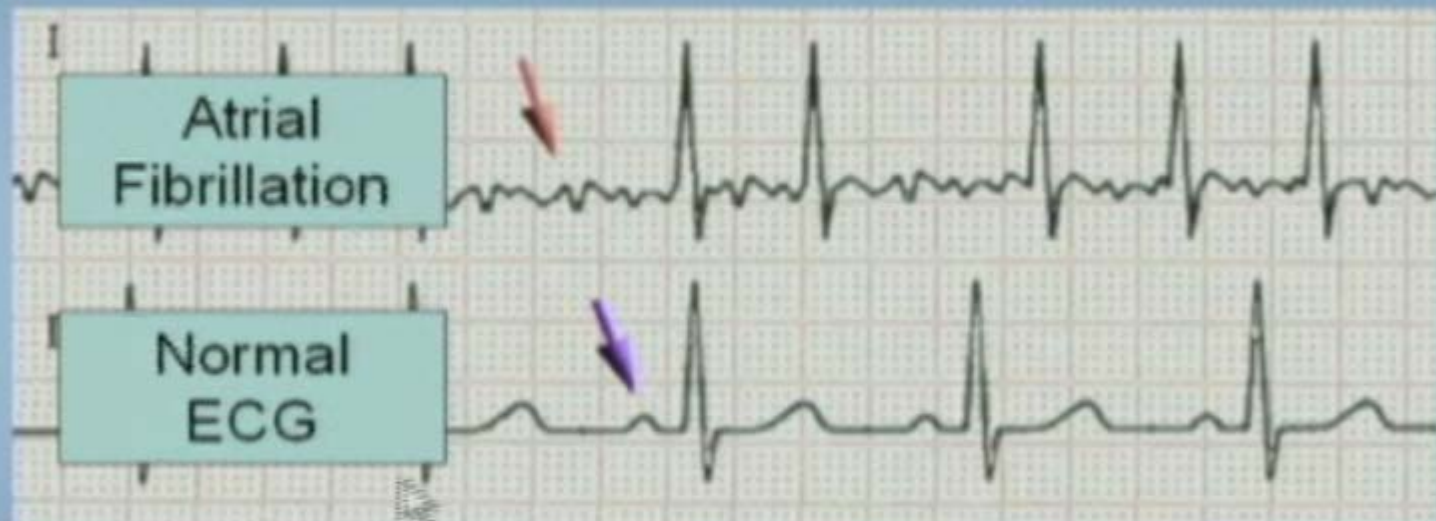
## Atrial Fibrillation (*cont'd.*)



## Atrial Fibrillation (cont'd.)

- ***Evaluation of patients with A-fib:***
  1. Is this the first episode?
  2. Is the patient symptomatic?
  3. Is the disease self-limited?
- ***Regardless of symptomatology, the risk of a thromboembolic event is equal, whether symptomatic or not, if the A-fib persists!!!***

## Atrial Fibrillation (*cont'd.*)



This image was reproduced from Wikipedia,  
<http://www.wikipedia.com>

## Atrial Fibrillation (*cont'd.*)

- **Management of A-fib:**
  1. Rhythm control versus rate control
    - RACE trial and AFFRIM trial
  2. In general:
    - Rate control is applied to the asymptomatic or those with little discomfort.
    - Rhythm control is applied to those with immediate symptoms or those who are unstable.

## Atrial Fibrillation (*cont'd.*)

- **Ventricular Rate Control:**
  1. Goal is <100 bpm
  2. Does not convert the rhythm but aids to stabilize the patient acutely
  3.  $\beta$ -blockers  $\rightarrow$  atenolol, metoprolol
  4. CCAs  $\rightarrow$  verapamil, diltiazem
  5. Beta blockers or Digoxin are the drugs of choice in those with CHF

## Atrial Fibrillation (*cont'd.*)

- *Rhythm Control:*

1. *Mechanical Cardioversion:*

- Synchronized electric shock
- Can be performed electively or emergently
- If performing electively → ***anticoagulate the patient!!!***



# Atrial Fibrillation (cont'd.)

- Rhythm Control (cont'd.):

- 2. Pharmacologic Cardioversion:

- Less effective than synchronized cardioversion
    - Anticoagulation
    - Drugs: Amiodarone, dofetilide, flecainide, ibutilide, propafenone, and quinidine
    - Drugs to maintain sinus rhythm:
      - » Amiodarone, disopyramide, dofetilide, flecainide, propafenone, and sotalol



## Atrial Fibrillation (*cont'd.*)

- **Newer treatment techniques:**

1. Catheter ablation

- Foci from pulmonary veins most common
- Called the Maze procedure

- **Other key points about A-fib:**

1. Rate control and chronic anticoagulation is the recommended treatment for all patients with chronic A-fib.

## Clinical Cases

A 32-year-old male presents with shortness of breath. His BP in the emergency room is 80/55 mm Hg. He has JVD and bilateral crackles upon physical examination. His ECG shows evidence of A-fib. What is the next best step in the management of this patient?

***Electric cardioversion***

## WPW (*cont'd.*)

- **Management:**

1. Acute:

- Unstable patient → immediate synchronized cardioversion
- Stable patient → procainamide (drug of choice)
  - Avoid digoxin,  $\beta$ -blockers, and CCAAs

## WPW (*cont'd.*)

- **Management:**

1. Acute:

- Unstable patient → immediate synchronized cardioversion
- Stable patient → procainamide (drug of choice)
  - Avoid digoxin,  $\beta$ -blockers, and CCAAs

2. Long-term:

- Ablation

# Ventricular Tachycardia (V-tach)

Defined as three or more consecutive ventricular beats at  $>120$  bpm

***Wide, bizarre QRS complexes***

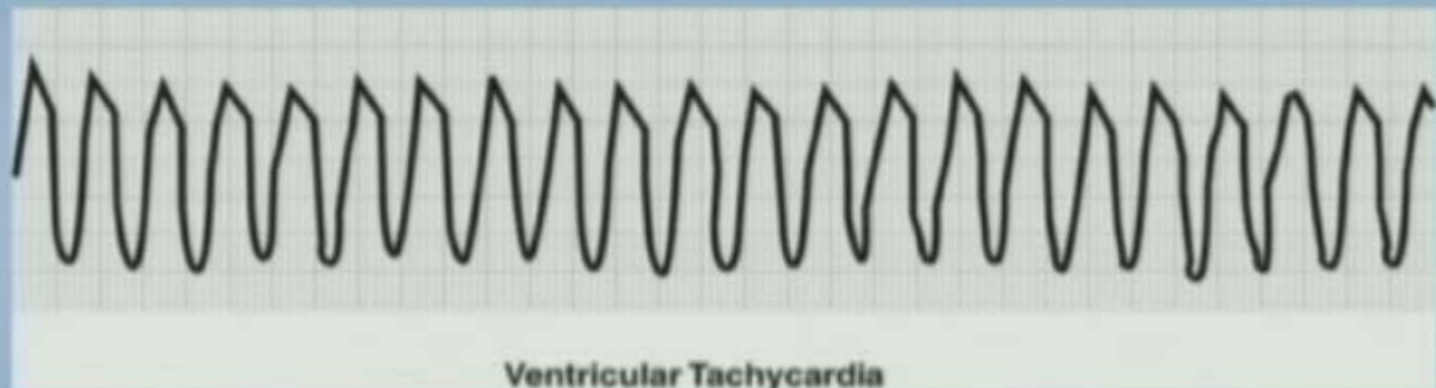
- **Etiology:**

1. Ischemic heart disease, especially post-MI
2. Cardiomyopathies
3. Metabolic derangements

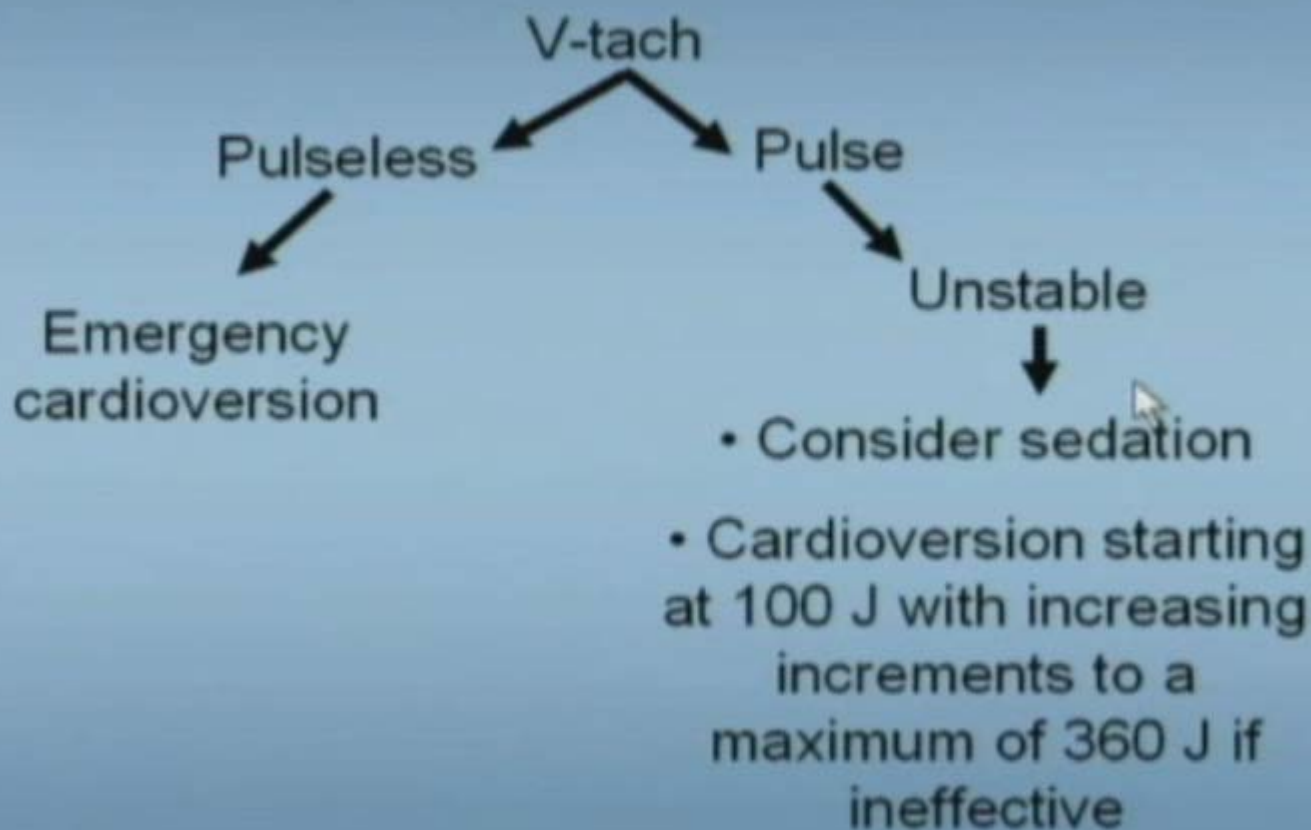




## V-tach (*cont'd.*)



# Management of V-tach



# Management of V-tach (*cont'd.*)

V-tach



Pulse and hemodynamically stable



- Oxygen

- Amiodarone or lidocaine 1 mg/kg loading dose



Lidocaine 0.5-3 mg/kg q 8 minutes until resolution



Procainamide 20-100 mg/kg until resolution



## Torsades de Pointes

- Undulating rotations of the QRS complex around the ECG baseline
  1. Premature ventricular beat
  2. Abnormal ventricular repolarization
  3. Prolonged QT-interval

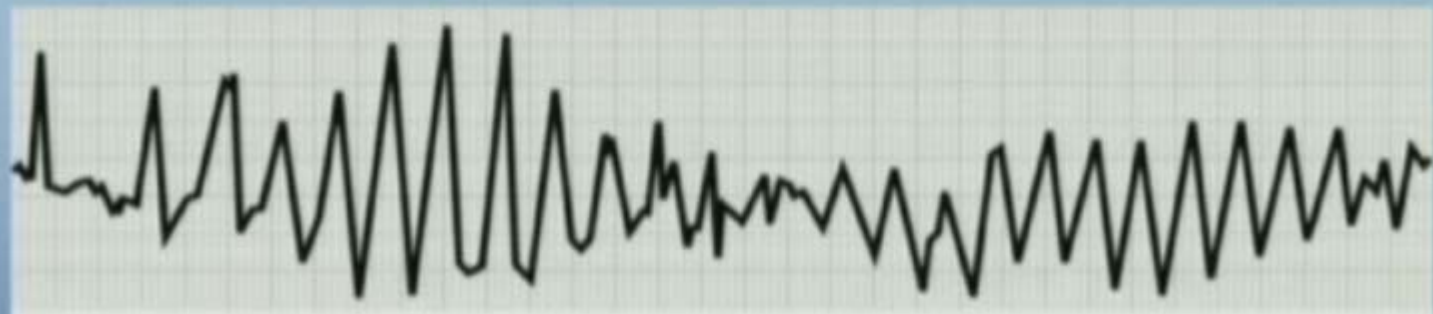


Figure I-5-4. Torsade